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NEWS 4	Apr 09	ZDB will be removed from STN
NEWS 5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
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NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
NEWS 14	Jul 29	Enhanced polymer searching in REGISTRY
NEWS 15	Jul 30	NETFIRST to be removed from STN
NEWS 16	Aug 08	CANCERLIT reload
NEWS 17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18	Aug 08	NTIS has been reloaded and enhanced
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NEWS 20	Aug 19	IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22	Aug 26	Sequence searching in REGISTRY enhanced
NEWS 23	Sep 03	JAPIO has been reloaded and enhanced
NEWS 24	Sep 16	Experimental properties added to the REGISTRY file
NEWS 25	Sep 16	CA Section Thesaurus available in CAPLUS and CA
NEWS 26	Oct 01	CASREACT Enriched with Reactions from 1907 to 1985
NEWS 27	Oct 21	EVENTLINE has been reloaded
NEWS 28	Oct 24	BEILSTEIN adds new search fields
NEWS 29	Oct 24	Nutraceuticals International (NUTRACEUT) now available on STN
NEWS 30	Oct 25	MEDLINE SDI run of October 8, 2002
NEWS 31	Nov 18	DKILIT has been renamed APOLLIT
NEWS 32	Nov 25	More calculated properties added to REGISTRY
NEWS 33	Dec 02	TIBKAT will be removed from STN
NEWS 34	Dec 04	CSA files on STN
NEWS 35	Dec 17	PCTFULL now covers WP/PCT Applications from 1978 to date
NEWS 36	Dec 17	TOXCENTER enhanced with additional content
NEWS 37	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS 38	Dec 30	ISMEC no longer available
NEWS 39	Jan 13	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 40	Jan 21	NUTRACEUT offering one free connect hour in February 2003
NEWS 41	Jan 21	PHARMAML offering one free connect hour in February 2003
NEWS 42	Jan 29	Simultaneous left and right truncation added to COMPENDEX,

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ENERGY, INSPEC
NEWS 43 Feb 13 CANCERLIT is no longer being updated
NEWS 44 Feb 24 METADEX enhancements
NEWS 45 Feb 24 PCTGEN now available on STN
NEWS 46 Feb 24 TEMA now available on STN
NEWS 47 Feb 26 NTIS now allows simultaneous left and right truncation
NEWS 48 Feb 26 PCTFULL now contains images
NEWS 49 Mar 04 SDI PACKAGE for monthly delivery of multifile SDI results

NEWS EXPRESS January 6 CURRENT WINDOWS VERSION IS V6.01a,
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AND CURRENT DISCOVER FILE IS DATED 01 OCTOBER 2002
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=> s zn glycoprotein
L1 9 ZN GLYCOPROTEIN

=> s zn and glycoprotein
L2 995 ZN AND GLYCOPROTEIN

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=> s l2 and (lipid or lipolytic)
L3          52 L2 AND (LIPID OR LIPOLYTIC)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4           34 DUP REM L3 (18 DUPLICATES REMOVED)

=> s l2 and (lipid or lipolytic or lpf)
L5          52 L2 AND (LIPID OR LIPOLYTIC OR LPF)

=> s l4 and py<=1998
   1 FILES SEARCHED...
   4 FILES SEARCHED...
L6          21 L4 AND PY<=1998

=> d l6 1-21 bib hit

L6   ANSWER 1 OF 21  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN   1998:306053  BIOSIS
DN   PREV199800306053
TI   Purification and characterization of a tumor lipid-mobilizing
     factor.
AU   Todorov, Penio; McDevitt, Trudi M.; Meyer, David J.; Ueyama, Hisao;
     Ohkubo, Iwao; Tisdale, Michael J. (1)
CS   (1) Pharmaceutical Sci. Inst., Aston Univ., Birmingham B4 7ET UK
SO   Cancer Research, (June 1, 1998) Vol. 58, No. 11, pp. 2353-2358.
     ISSN: 0008-5472.
DT   Article
LA   English
TI   Purification and characterization of a tumor lipid-mobilizing
     factor.
SO   Cancer Research, (June 1, 1998) Vol. 58, No. 11, pp. 2353-2358.
     ISSN: 0008-5472.
AB   Cancer patients with weight loss showed urinary excretion of a
     lipid-mobilizing factor (LMF), determined by the ability to
     stimulate lipolysis in isolated murine epididymal adipocytes. Such
     bioactivity was not detectable in the urine of cancer patients without
     weight loss or in normal subjects. The LMF was purified using a
     combination of ion exchange, exclusion, and hydrophobic interaction
     chromatographies to give a single component of apparent M. 43,000, which
     showed homology in amino acid sequence with human plasma Zn
     -alpha2-glycoprotein. Both substances showed the same mobility
     on denaturing and nondenaturing gels and the same chymotrypsin digestion
     pattern, both stained heavily for carbohydrate, and they showed similar
     immunoreactivity. Polyclonal antisera to human plasma Zn-alpha2-
     glycoprotein was also capable of neutralization of the bioactivity
     of human LMF in vitro. Using competitive PCR to quantify expression of
     Zn-alpha2-glycoprotein, we found that only those tumors
     that were capable of producing a decrease in carcass lipid
     expressed mRNA for Zn-alpha2-glycoprotein. These
     results provide strong evidence to suggest that tumor production of
     Zn-alpha2-glycoprotein is responsible for the
     lipid catabolism seen in cancer patients.
IT   Major Concepts
        Tumor Biology
IT   Diseases
        cancer: neoplastic disease
IT   Chemicals & Biochemicals
        mRNA [messenger RNA]: expression; tumor lipid-mobilizing
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factor: characterization, purification; zinc-alpha-2-**glycoprotein**

IT Miscellaneous Descriptors
lipid catabolism

L6 ANSWER 2 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1998:296721 BIOSIS
DN PREV199800296721
TI Biological evaluation of a **lipid**-mobilizing factor isolated from the urine of cancer patients.
AU Hirai, Kouzo; Hussey, Helen J.; Barber, Matthew D.; Price, Sarah A.; Tisdale, Michael J. (1)
CS (1) Pharmaceutical Sci. Inst., Aston Univ., Birmingham B4 7ET UK
SO Cancer Research, (**June 1, 1998**) Vol. 58, No. 11, pp. 2359-2365. ISSN: 0008-5472.
DT Article
LA English
TI Biological evaluation of a **lipid**-mobilizing factor isolated from the urine of cancer patients.
SO Cancer Research, (**June 1, 1998**) Vol. 58, No. 11, pp. 2359-2365. ISSN: 0008-5472.
AB We have previously shown human **lipid**-mobilizing factor (LMF) to be homologous with the plasma protein **Zn**-alpha2-**glycoprotein** in amino acid sequence, electrophoretic mobility, and immunoreactivity. In this study, both LMF and **Zn**-alpha2-**glycoprotein** have been shown to stimulate glycerol release from isolated murine epididymal adipocytes with a comparable dose-response profile. Both LMF and **Zn**-alpha2-**glycoprotein** caused a stimulation of adenylate cyclase in murine adipocyte plasma membranes in a GTP-dependent process, with maximum stimulation at 0.1 muM GTP and with saturation at protein concentrations of >5 mug/assay. Administration of LMF to exbreeder male mice over a 89-h period produced a decrease in body weight without a change in food and water intake. Body composition analysis showed a 42% reduction in carcass **lipid** when compared with controls. Treatment of ob/ob mice with human LMF over a 160-h period also produced a decrease in body weight, with a 19% reduction in carcass fat, without a change in body water or nonfat mass. Serum levels of glycerol and 3-hydroxybutyrate were significantly increased, as was oxygen uptake by interscapular brown adipose tissue, providing evidence of increased **lipid** mobilization and utilization. Human white adipocytes responded to both LMF and isoprenaline to the same extent, although the maximal response was lower than that for murine white adipocytes. These results suggest that LMF not only has the capacity to induce **lipid** mobilization and catabolism in mice, but it also has the potential to exert similar effects in cachectic cancer patients.
IT Major Concepts
Biochemistry and Molecular Biophysics; Tumor Biology
IT Parts, Structures, & Systems of Organisms
epididymal adipocytes; urine: excretory system
IT Diseases
cancer: neoplastic disease
IT Chemicals & Biochemicals
adenylate cyclase; glycerol: release; **lipid**-mobilizing factor: evaluation; zinc-alpha-2-**glycoprotein**
IT Miscellaneous Descriptors
body composition; **lipid** catabolism

L6 ANSWER 3 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1993:497113 BIOSIS
DN PREV199396121120

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TI Binding of cerebrosides and sulfatides to saposins A-D.
AU Soeda, Shinji; Hiraiwa, Masao; O'Brien, John S.; Kishimoto, Yasuo (1)
CS (1) Cent. Mol. Genetics, 0634J, Univ. Calif. San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0634 USA
SO Journal of Biological Chemistry, (1993) Vol. 268, No. 25, pp. 18519-18523. ISSN: 0021-9258.
DT Article
LA English
SO Journal of Biological Chemistry, (1993) Vol. 268, No. 25, pp. 18519-18523. ISSN: 0021-9258.
AB Saposins are a family of four small **glycoproteins**, all of which are derived from prosaposin, and are involved in the lysosomal hydrolysis of various sphingolipids. Results from this investigation demonstrate that saposins A-D bind to galactosyl- and glucosylceramide. The binding was highly dependent on the solution pH; maximum binding of glucosylceramide to all saposins occurred at pH 7. Maximum binding of galactosylceramide to saposins B and D occurred at a more basic pH (8.5). The binding of glucosylceramide to saposins was significantly inhibited by Mg-2+, Ca-2+, or **Zn**-2+. Although maximum binding of sulfatide to saposins A, C, and D occurred at acidic pH, the binding to saposin B was maximum at pH 8.5. Saposin A also bound sphingomyelin or phosphatidylcholine at neutral pH. No significant binding was evident between these **lipids** and saposins B-D at any pH value. The existence of saposin-**lipid** complexes was further confirmed in selected samples by gel filtration, isoelectric focusing, and a TLC binding assay. We have also shown that galactosylceramide bound to saposins A-D was efficiently transported to a rat brain microsomal fraction. This result suggests that saposins and possibly their precursor, prosaposin, may be involved in membrane biogenesis such as the assembly of myelin and plasma membranes.

L6 ANSWER 4 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1982:287046 BIOSIS
DN BA74:59526
TI ZINC IODIDE OSMIUM STAINING OF MEMBRANE COATING GRANULES IN KERATINIZED AND NONKERATINIZED MAMMALIAN ORAL EPITHELIUM.
AU SQUIER C A
CS DEP. ORAL PATHOL., UNIV. IOWA, IOWA CITY, IOWA 52242, USA.
SO ARCH ORAL BIOL, (1982) 27 (5), 377-382. CODEN: AOBIAR. ISSN: 0003-9969.
FS BA; OLD
LA English
SO ARCH ORAL BIOL, (1982) 27 (5), 377-382. CODEN: AOBIAR. ISSN: 0003-9969.
AB Specimens of keratinized and nonkeratinized oral epithelium [from rats, rabbits and monkeys] were examined in the EM after being stained with **Zn** iodide-osmium. In both types of tissue, reaction was seen in unmyelinated nerves, in the specific granules of epithelial Langerhans cells and within lysosome-like organelles and small vesicles associated with Golgi systems. In keratinized epithelia, the reaction was also present in the membrane-coating granules and between the deepest cells of the keratinized layer. In contrast, the membrane-coating granules of nonkeratinized epithelia lacked **Zn** iodide-osmium staining despite the presence of reaction in adjacent Golgi systems. **Zn** iodide-osmium probably stains glycolipid or **glycoprotein** material in the cell. This material is elaborated in the Golgi systems from which lysosomes and the membrane-coating granules of keratinized tissues are probably derived.
IT Miscellaneous Descriptors
RAT RABBIT MONKEY UNMYELINATED NERVE LANGERHANS CELL GLYCO **LIPID** GLYCO PROTEIN

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L6 ANSWER 5 OF 21 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1977:209793 BIOSIS
DN BA64:32157
TI PURIFICATION AND CHARACTERIZATION OF ALPHA-D MANNOSIDASE EC-3.2.1.24 FROM RAT LIVER GOLGI MEMBRANES.
AU TULSIANI D R P; OPHEIM D J; TOUSTER O
SO J BIOL CHEM, (1977) 252 (10), 3227-3233.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA Unavailable
SO J BIOL CHEM, (1977) 252 (10), 3227-3233.
CODEN: JBCHA3. ISSN: 0021-9258.
AB Rat liver contains 3 .alpha.-D-mannosidases [EC 3.2.1.24] occurring in different intracellular fractions. The present paper reports the isolation of the mannosidase of Golgi membranes, in which the enzyme is a distinctive glycosidase component. The Golgi mannosidase was extracted with detergent and purified to apparent homogeneity, all solutions requiring the presence of detergent (0.1% Triton X-100) to maintain the enzyme in soluble form. In molecular weight determinations gel chromatography on Sephadex G-200 yielded a value of 295,000, whereas sucrose density gradient centrifugation gave a value of 110,000. Under dissociating conditions, polyacrylamide gel electrophoresis showed 2 bands, corresponding to MW of 75,000-80,000 and 145,000-150,000. The mannosidase may be a tetrameric protein of approximately 300,000 MW, and that the dimeric form is relatively stable. The pH optimum is 5.5; the isoelectric point is 5.8. Since the enzyme stains for carbohydrate (but not for **lipid**) and binds to concanavalin A, it is presumably a **glycoprotein**. Although chelating agents have no effect on enzyme activity, **Zn** and Co cations, as well as sulfhydryl compounds, are activators. Since the properties of the purified Golgi .alpha.-D-mannosidase differ so greatly from those of the lysosomal and cytosolic .alpha.-D-mannosidase, it is unlikely to be biosynthetically related to the latter enzymes and undoubtedly has a distinctive function in Golgi membranes, presumably in glycopolymer metabolism.

L6 ANSWER 6 OF 21 MEDLINE
AN 93346762 MEDLINE
DN 93346762 PubMed ID: 8345200
TI Clusterin, the human apolipoprotein and complement inhibitor, binds to complement C7, C8 beta, and the b domain of C9.
AU Tschopp J; Chonn A; Hertig S; French L E
CS Institute of Biochemistry, University of Lausanne, Epalinges, Switzerland.
SO JOURNAL OF IMMUNOLOGY, **(1993 Aug 15)** 151 (4) 2159-65.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199309
ED Entered STN: 19930924
Last Updated on STN: 19950206
Entered Medline: 19930908
SO JOURNAL OF IMMUNOLOGY, **(1993 Aug 15)** 151 (4) 2159-65.
Journal code: 2985117R. ISSN: 0022-1767.
AB Clusterin is a heterodimeric multifunctional protein expressed in a variety of tissues and cells. It forms high density **lipid** complexes in plasma and participates in the control of the lytic activity of the late complement complex (TCC, C5b-9). Together with vitronectin, clusterin binds to the nascent amphiphilic C5b-9 complex, rendering it

water soluble and lytically inactive. To define the interactions that underlie the complement-inhibitory function of clusterin, we have examined the binding interactions between [125I]clusterin and the isolated components of the complex, C5b-6, C7, C8, and C9 and vitronectin. By using ligand blotting in the presence of Tween, specific binding of the labeled clusterin with C7, the beta-subunit of C8 and C9 was detected. Binding to C9 was competed by polymerized C9, but not by C8, C7, C6, and CD59, suggesting that the conformational change occurring during the hydrophilic-amphiphilic transition of C9 exposes the interaction site for clusterin. When thrombin-treated C9 was analyzed, clusterin was found to recognize the C9b fragment containing the hydrophobic membrane interaction segment. Both subunits of clusterin interact with C9 and are similarly potent in inhibiting C5b-9-mediated hemolysis and **Zn**+(+)-induced C9 polymerization. These results show that clusterin exerts its inhibitory effect by interacting with a structural motif common to C7, C8 alpha, and C9b.

CT Check Tags: Human; In Vitro; Support, Non-U.S. Gov't
*Complement 7: ME, metabolism
*Complement 8: ME, metabolism
*Complement 9: ME, metabolism
***Glycoproteins: ME, metabolism**
Glycoproteins: PD, pharmacology
Hemolysis: DE, drug effects
Peptide Fragments: ME, metabolism
Protein Binding

CN 0 (Complement 7); 0 (Complement 8); 0 (Complement 9); 0 (**Glycoproteins**); 0 (Peptide Fragments); 0 (clusterin)

L6 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1998:757058 CAPLUS
DN 130:137680
TI Peroxidative reactions in the vitreous as related to diabetic retinopathy
AU Tanaka, Yasushi
CS Dep. Ophthalmol., Koshigaya Hosp., Dokkyo Univ. Sch. Med., Saitama, 343-8555, Japan
SO Nippon Ganka Gakkai Zasshi (**1998**), 102(9), 576-582
CODEN: NGZAA6; ISSN: 0029-0203
PB Nippon Ganka Gakkai
DT Journal
LA Japanese
SO Nippon Ganka Gakkai Zasshi (**1998**), 102(9), 576-582
CODEN: NGZAA6; ISSN: 0029-0203
AB The blood of diabetics often shows enhanced peroxidative reactions and non-enzymic glycosylation, or glycation. These features should also be manifest in the vitreous in diabetic eyes. I quantitated the level of superoxide dismutase (SOD) in the serum and the vitreous in 22 eyes of 23 diabetics and in 16 eyes of 16 nondiabetics. The total amt. of serum SOD was the same in both groups. There was a significant decrease in SOD activity in the diabetic vitreous ($p<0.05$). The diabetic vitreous also showed increases in glycated Cu, **Zn**-SOD and glycated protein. The level of **lipid** peroxidases was significantly increased in the diabetic vitreous ($P<0.05$). These findings suggest that glycation is enhanced in the diabetic vitreous resulting in collapse of active oxygen scavenging and in progressed peroxidn.

IT **Glycoproteins**, general, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(peroxidative reactions in vitreous as related to diabetic retinopathy in humans)

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L6 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1997:564491 CAPLUS
DN 127:219967
TI Evaluation of oxidative stress and protection by antioxidants in Moroccan malnourished children
AU Squali Houssaini, Fatima Zahra; Arnaud, Josiane; Richard, Marie Jeanne; Renversez, Jean Charles; Favier, Alain
CS Faculte Sciences Dhar Mehraz, Universite Sidi Med Ben, Fes, Morocco
SO Annals of Nutrition & Metabolism (**1997**), 41(3), 149-159
CODEN: ANUMDS; ISSN: 0250-6807
PB Karger
DT Journal
LA French
SO Annals of Nutrition & Metabolism (**1997**), 41(3), 149-159
CODEN: ANUMDS; ISSN: 0250-6807
AB In morocco, malnutrition is a public health problem. Indeed, 25% of 6-60-mo-old children suffer from malnutrition. Imbalance between antioxidant protection and prooxidant stress was reported to accurately predict the survival of malnourished children. Therefore, we detd. blood antioxidant vitamins (retinol, .alpha.-tocopherol and carotenoids), trace elements (serum **Zn**, Cu, and Se) and enzymes (erythrocyte Se glutathione peroxydase and Cu-**Zn** superoxide dismutase) as well as blood oxidative stress index [ferritin, thiobarbituric acid-reactive substances (TBARS)] in 21 children suffering from severe malnutrition, 15 children suffering from mild malnutrition and in 20 healthy control children. Se, retinol, .alpha.-tocopherol, and carotenoids were significantly decreased in malnourished children. These decreases were related to the severity of malnutrition. Moreover, the percentage of vitamin and trace element concns. under deficient cutoff were high in malnourished children. On the contrary, TBARS, ferritin and prognostic inflammatory and nutritional index (PINI) were significantly increased in malnourished children. Except for TBARS, these increases were related to the severity of malnutrition. On the other hand, blood retinol, .alpha.-tocopherol, .beta.-carotene, and Se were neg. related to .alpha.1-acid **glycoprotein**. Blood .beta.-cryptoxanthin, lycopene, carotenes, and Cu were pos. related to wt. Finally, blood lutein/zeaxanthin and Cu were pos. related to height. These results confirm the imbalance between antioxidant protective factors and oxidative stress index in malnourished children. Moreover, the decrease in antioxidant protective factors is related to inflammation or stature. These results suggest that antioxidant micronutrient supplementation of the refeeding diet could be required in the nutritional rehabilitation of malnourished children.
IT Peroxidation
(**lipid**, TBARS, antioxidant; evaluation of oxidative stress and protection by antioxidants in Morocean malnourished children)
IT **Lipids**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(peroxidn., TBARS, antioxidant; evaluation of oxidative stress and protection by antioxidants in Morocean malnourished children)

L6 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1997:154172 CAPLUS
DN 126:210548
TI Oxidative stress caused by glycation of Cu,**Zn**-superoxide dismutase and its effects on intracellular components
AU Fujii, Junichi; Myint, Theingi; Okado, Ayako; Kaneto, Hideaki; Taniguchi, Naoyuki
CS Department of Biochemistry, Osaka University Medical School, Suita, 565,

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Japan
SO Nephrology, Dialysis, Transplantation (**1996**), 11(Suppl. 5, Advanced Glycation End-Products in Diabetes Mellitus and Renal Failure), 34-40
CODEN: NDTREA; ISSN: 0931-0509
PB Oxford University Press
DT Journal
LA English
TI Oxidative stress caused by glycation of Cu,**Zn**-superoxide dismutase and its effects on intracellular components
SO Nephrology, Dialysis, Transplantation (**1996**), 11(Suppl. 5, Advanced Glycation End-Products in Diabetes Mellitus and Renal Failure), 34-40
CODEN: NDTREA; ISSN: 0931-0509
AB It is now evident that the redox state of the cell is a pivotal determinant of the fate of cells. Extensive prodn. of reactive oxygen species (ROI) causes necrotic cell death. Even transient or localized prodn. of ROI may mediate a signal for apoptotic cell death, whereas small amts. of ROI function as an intracellular messenger of some growth stimulants. Accumulating evidence supports the concept that decrease in Cu,**Zn**-superoxide dismutase (SOD) activity causes apoptotic cell death in neuronal cells. Our data using mutant Cu,**Zn**-SOD related to familial amyotrophic lateral sclerosis (FALS) suggest that glycation itself and ROI produced from the glycated proteins are involved in many diseases, including diabetic complications. Glycation of important cellular components, including **lipid**, DNA and proteins, induces dysfunction of these components. Mutant proteins in patients with various hereditary diseases would be destabilized by the glycation reaction, as shown in the case of mutant Cu,**Zn**-SODs, thereby hyperglycemic conditions would trigger the onset of some hereditary diseases such as FALS and Alzheimer's disease. Glycation, particularly of antioxidative enzymes, would enhance prodn. of ROI, resulting in oxidative damage to the cells.
IT Nervous system
(familial amyotrophic lateral sclerosis; oxidative stress caused by glycation of Cu,**Zn**-superoxide dismutase and effects on intracellular components in relation to disease)
IT Alzheimer's disease
Apoptosis
Diabetes mellitus
Glycation
Oxidative stress, biological
(oxidative stress caused by glycation of Cu,**Zn**-superoxide dismutase and effects on intracellular components in relation to disease)
IT **Glycoproteins**, general, biological studies
Reactive oxygen species
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PROC (Process)
(oxidative stress caused by glycation of Cu,**Zn**-superoxide dismutase and effects on intracellular components in relation to disease)
IT 9054-89-1
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(copper-zinc-contg.; oxidative stress caused by glycation of Cu, **Zn**-superoxide dismutase and effects on intracellular components

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in relation to disease)

IT 7782-44-7D, Oxygen, radicals, biological studies
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PROC (Process)
(oxidative stress caused by glycation of Cu,**Zn**-superoxide dismutase and effects on intracellular components in relation to disease)

L6 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1996:618396 CAPLUS
DN 126:5163
TI Correlation between blood plasma transferrin and liver malonaldehyde in mice
AU Kon, I. Ya.; Shilina, N. M.; Koterov, A. N.
CS Institut Pitaniya, Moscow, 109240, Russia
SO Biokhimiya (Moscow) (**1996**), 61(7), 1198-1203
CODEN: BIOHAO; ISSN: 0320-9725
PB Nauka
DT Journal
LA Russian
SO Biokhimiya (Moscow) (**1996**), 61(7), 1198-1203
CODEN: BIOHAO; ISSN: 0320-9725
AB The plasma transferrin content and the level of 2-thiobarbituric acid-reactive substances, malonaldehyde (MDA), in mouse liver were assayed under the induction or the inhibition of **lipid** peroxidn. with bromobenzene (BB) or **Zn**-metallothionein (**Zn**-MT), resp. Blood transferrin concn. decreased and MDA level in liver increased after BB injection. On the contrary, blood transferrin concn. increased and liver MDA content decreased (two- to three-fold each) after **Zn** -MT injection. The effects of **Zn**-MT were obsd. even in BB-injected animals. **Zn**-MT injection increased total transferrin content in the blood and decreased glycosylation of the **glycoprotein**. Changes in blood transferrin content correlated neg. (r= -0.75) with the changes in liver MDA level. The data confirmed that plasma transferrin could participate in the regulation of tissue **lipid** peroxidn.
ST transferrin blood plasma malonaldehyde liver; **lipid** peroxidn transferrin blood plasma
IT **Lipids**, biological studies
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(peroxidn. products; correlation between blood plasma transferrin and liver malonaldehyde in mice)

L6 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1994:627925 CAPLUS
DN 121:227925
TI Solubilization of full-length amyloid precursor proteins from PC12 cell membranes
AU Ripellino, J. A.; Vassilacopoulou, D.; Robakis, N. K.
CS Mount Sinai School of Medicine, Department of Psychiatry, New York, NY, USA
SO Journal of Neuroscience Research (**1994**), 39(2), 211-18
CODEN: JNREDK; ISSN: 0360-4012
DT Journal
LA English
SO Journal of Neuroscience Research (**1994**), 39(2), 211-18

09921880

CODEN: JNREDK; ISSN: 0360-4012

AB The amyloid .beta. protein (A.beta.) of Alzheimer disease (AD) is derived from the proteolytic processing of the amyloid precursor proteins (APP), which are considered type I transmembrane proteins. Prodn. of A.beta. from a transmembrane precursor predicts a proteolytic cleavage within the **lipid** bilayer, a site relatively inaccessible to proteases. Here the authors show that incubation of a membrane fraction of PC12 cells at 37.degree. results in the solubilization of an APP species which migrates on SDS-PAGE as full-length APP. The release of this full-length APP was pH-dependent with a peak of activity of pH 9.0. At this pH about 19% of the membrane APP was released from the active subcellular fraction. Under the same conditions other transmembrane proteins remained insol. Very little APP was solubilized at 4.degree.. APP solubilization was specifically inhibited by the serine protease inhibitors aprotinin and pefabloc. Other protease inhibitors, including leupeptin and .alpha.1-antitrypsin, had no effect. Several metal cations, including Ca++ and **Zn**++, also inhibited release of sol. full-length aPP. Low levels of full-length APP were also detected in both the sol. fraction of PC12 cell exts. and in the media of PC12 cell cultures. These data suggest the involvement of a serine protease in the solubilization of membrane, full-length APP. The release of this APP could provide a sol. substrate for the proteolytic enzymes involved in the prodn. of A.beta..

IT **Glycoproteins**, specific or class
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(amyloid A4, pre-, solubilization of full-length amyloid precursor proteins from PC12 cell membranes, involvement of a serine protease in A.beta. formation after release of APP)

L6 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1990:401673 CAPLUS
DN 113:1673
TI Cell and molecular mechanisms of polymetallic dust effect on respiratory organs
AU Burkhanov, A. I.; Bazelyuk, L. T.
CS Karaganda. Pedagog. Inst., Karaganda, USSR
SO Gigiena i Sanitariya (**1990**), (3), 15-17
CODEN: GISAAA; ISSN: 0016-9900
DT Journal
LA Russian
SO Gigiena i Sanitariya (**1990**), (3), 15-17
CODEN: GISAAA; ISSN: 0016-9900
AB Cytochem. indicators of metabolic processes in alveolar macrophage of rats following intratracheal administration of Pb-**Zn** conc. (Pb 50, **Zn** 15, As 8, Se 1, SiO2 1%, etc.) are described. The Pb-**Zn** conc. disrupted protein metab., decreased nucleoproteins, neutral **lipid** and phospholipid levels aerobic and anaerobic oxidn. marker enzymes, etc. Disturbance in the metabolic processes of the mononuclear phagocytes led to their death. The Pb-**Zn** conc. was toxic at both the cellular and mol. level.

IT Carbohydrates and Sugars, biological studies
Glycoproteins, biological studies
Lipids, biological studies
Phospholipids, biological studies
Proteins, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metab. of, lead-zinc conc. effect on)

09921880

L6 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1985:468951 CAPLUS
DN 103:68951
TI Zinc-induced platelet aggregation is mediated by the fibrinogen receptor and is not accompanied by release or by thromboxane synthesis
AU Heynes, Anthon du P.; Eldor, Amiram; Yarom, Rena; Marx, Gerard
CS Dep. Hematol., Hadassah Univ. Hosp., Jerusalem, 91120, Israel
SO Blood (**1985**), 66(1), 213-19
CODEN: BLOOAW; ISSN: 0006-4971
DT Journal
LA English
SO Blood (**1985**), 66(1), 213-19
CODEN: BLOOAW; ISSN: 0006-4971
AB **Zn** (0.1-0.3 mM) induces aggregation of washed human platelet suspensions. Higher concns. (1-3 mM) of **Zn** were needed to aggregate platelets in platelet-rich plasma obtained from blood anticoagulated with low-mol.-wt. heparin, probably due to the binding of **Zn** to the plasma proteins. **Zn**-induced aggregation of normal washed platelets required added fibrinogen and no aggregation occurred with thrombasthenic platelets or with normal platelets pretreated with a monoclonal antibody (10E5) that blocks the platelet fibrinogen receptor. Apparently the platelet membrane fibrinogen receptor-**glycoproteins** IIb and IIIa mediate the effect of **Zn**. **Zn**-induced aggregation was blocked by the agent TMB-8, which interferes with the internal Ca2+ flux, and by prostacyclin, which elevates platelet cAMP levels. **Zn**-induced aggregation was not accompanied by thromboxanes synthesis or by the secretion of dense-body serotonin and was not affected by preexposure of platelets to acetylsalicylic acid. Expts. with creatine phosphate/creatine phosphokinase showed that the **Zn** effect on platelets was independent of extracellular ADP. **Zn** had an additive effect when platelet aggregation was stimulated with subthreshold concns. of collagen or ADP. Together with the known effects of nutritional **Zn** on in vivo bleeding, platelet aggregation, and **lipid** metab., the results suggest that **Zn** may have an important bearing on normal hemostasis, thrombosis, and atherosclerosis.
IT **Glycoproteins**
RL: BIOL (Biological study)
(IIIa, blood platelet of human in zinc-induced aggregation in relation to)
IT **Glycoproteins**
RL: BIOL (Biological study)
(IIb, blood platelet of human in zinc-induced aggregation in relation to)

L6 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1984:152076 CAPLUS
DN 100:152076
TI Comparative characteristics of the effects of hydrogen fluoride and hydrogen phosphide with varying activity levels on animals
AU Atchabarov, B. A.; Aitbaev, T. Kh.; Aitbembetov, B. N.
CS Kaz. Nauchno-Issled. Inst. Kraevoi Patol., Alma-Ata, USSR
SO Zdravookhranenie Kazakhstana (**1984**), (1), 28-31
CODEN: ZDKAA8; ISSN: 0372-8277
DT Journal
LA Russian
SO Zdravookhranenie Kazakhstana (**1984**), (1), 28-31
CODEN: ZDKAA8; ISSN: 0372-8277
AB The chronic exposure of HF and PH3 (administered as **Zn** phosphide which under gastric HCl forms PH3) caused similar changes in a no. of

biochem. indicators. Hepatic changes e.g., in hippuric acid, glycogen, **lipids**, succinate dehydrogenase, sugar, and in blood **glycoproteins** were noted. The differences in some changes included the blood proteins under HF and cholinesterase, sialic acids, and seromucoids under PH3. The changes seen under 5-fold max. permissible concn. (5 MPC) of HF and 0.5 MPC were identical and also those seen under 10 MPC HF and 0.1 MPC PH3.

L6 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1973:415686 CAPLUS
DN 79:15686
TI Action of proteolytic enzymes of Clostridium histolyticum and Clostridium novyi on human plasma proteins
AU Schallehn, Gisela; Mueller, Hans E.
CS Inst. Med. Mikrobiol. Immunol., Univ. Bonn, Bonn, Fed. Rep. Ger.
SO Zentralblatt fuer Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene, Abteilung 1: Originale, Reihe A: Medizinische Mikrobiologie und Parasitologie (**1973**), 224(1), 102-14
CODEN: ZMMPAO; ISSN: 0300-9688
DT Journal
LA German
SO Zentralblatt fuer Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene, Abteilung 1: Originale, Reihe A: Medizinische Mikrobiologie und Parasitologie (**1973**), 224(1), 102-14
CODEN: ZMMPAO; ISSN: 0300-9688
AB The proteolytic activity of C. histolyticum and C. novyi was studied by immunoelectrophoresis. The following human proteins were used as substrates: prealbumin, albumin, .alpha.1-lipoprotein, .alpha.1-acid **glycoprotein**, .alpha.1-antitrypsin, .alpha.1-antichymotrypsin, .alpha.1B-**glycoprotein**, .alpha.1T-**glycoprotein**, inter-.alpha.-trypsin inhibitor, haptoglobin, ceruloplasmin, C1s-inactivator, .alpha.2-macroglobulin, .alpha.2HS-**glycoprotein**, **Zn**-.alpha.2-**glycoprotein**, .beta.-lipoprotein, transferrin, .beta.1c/.beta.1A-globulin, hemopexin, fibrinogen, .beta.2-**glycoprotein**-I, IgA, IgM and IgG. Proteases of C. histolyticum were more active than that of C. novyi. C. novyi showed stronger **lipolytic** activity. The tissue lysis obsd. during C. histolyticum infection apparently results from the action of collagenase and elastase, rather than from nonspecific proteolysis. Therefore, the proteases of Cl. histolyticum and Cl. novyi are not comparable with those of Aeromonas hydrophila, Bacteroides melaninogenicus, Clostridium tetani, Proteus vulgaris, or Pseudomonas aeruginosa.

L6 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1972:561286 CAPLUS
DN 77:161286
TI Heterogeneity in the phospholipid content of purified rabies virus (ERA strain) particles
AU Sokol, Frantisek; Clark, H. F.; Gyorgy, Emese; Tomassini, Natale
CS Wistar Inst. Anat. Biol., Philadelphia, PA, USA
SO Journal of General Virology (**1972**), 16(2), 173-83
CODEN: JGVIAY; ISSN: 0022-1317
DT Journal
LA English
SO Journal of General Virology (**1972**), 16(2), 173-83
CODEN: JGVIAY; ISSN: 0022-1317
AB ERA strain rabies virus purified by **Zn** acetate pptn., Sephadex filtration, and sucrose d. gradient centrifugation lost a portion of its envelope phospholipids. High egg passage virus did not exhibit this behavior. The release of phospholipids from the rabies virus caused no

marked decrease in infectivity. The envelope changed, however, from bullet- to bag-shaped. The **glycoprotein** compn. and RNA suggest that any heterogeneity in sedimentation properties of enveloped **lipid**-contg. viruses should be interpreted cautiously.

L6 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2003 ACS
AN 1969:103319 CAPLUS
DN 70:103319
TI Inhibitory substances of gastric secretion. II. Extraction and separation of sialogastrone from human saliva
AU Kobayashi, Masayoshi; Yamamoto, Masaaki
CS Chem. Res. Lab., Teikoku Hormone Mfg. Co., Ltd., Kawasaki, Japan
SO Yakugaku Zasshi (**1969**), 89(2), 222-9
CODEN: YKKZAJ; ISSN: 0031-6903
DT Journal
LA Japanese
SO Yakugaku Zasshi (**1969**), 89(2), 222-9
CODEN: YKKZAJ; ISSN: 0031-6903
AB Human saliva was sepd. into dialyzable and nondialyzable fractions; the nondialyzable fraction powerfully inhibited gastric acid secretion and gastric ulceration. This active substance, designated sialogastrone (I), was purified by sequential **Zn** complex pptn., Me2CO fractionation, DEAE-cellulose column chromatog., and gel filtration. The purified I had an inhibitory activity 300 times that of the original lyophilized saliva. This prepn. was almost homogeneous by polyacrylamide-gel electrophoresis and had an electrophoretic mobility different from that of other neutral substances in saliva. This purified I contained 58.6% reducing sugars and 31.5% protein, but no P or **lipids**. Its mol. wt. was >150,000, as detd. by gel filtration.
ST sialogastrone isolation saliva; saliva sialogastrone isolation; gastric secretion inhibition sialogastrone; **glycoproteins** sialogastrone

L6 ANSWER 18 OF 21 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
AN 75007666 EMBASE
DN 1975007666
TI Chemical composition, affinity for calcium, and some related properties of the vitamin D dependent calcium binding protein.
AU Bredderman P.J.; Wasserman R.H.
CS Dept. Phys. Biol., New York State Veter. Coll., Cornell Univ., Ithaca, N.Y. 14850, United States
SO Biochemistry, (1974) 13/8 (1687-1694).
CODEN: BICHAW
DT Journal
FS 037 Drug Literature Index
002 Physiology
029 Clinical Biochemistry
030 Pharmacology
LA English
SO Biochemistry, (1974) 13/8 (1687-1694).
CODEN: BICHAW
AB The concentration of a vitamin D dependent intestinal high affinity Ca binding protein (CaBP) is known to be highly correlated with the vitamin D dependent enhancement of intestinal Ca absorption. Purified CaBP from chick duodenal mucosa was analyzed for **lipids**, **glycoprotein** carbohydrate components, amino acid composition, and Ca binding properties. It was free of **lipid**, carbohydrate, phosphorus, and other ash producing substances. The molecular weight from amino acid composition and sodium dodecyl sulfate polyacrylamide gel electrophoresis was near 28,000. CaBP contains only three half cystine residues. Several spectrophotometric methods, including a new three

wavelength method, indicated the presence of 2 tryptophan residues. Polar residues make up 53% of the 242 residues and 61 residues contain side chain carboxyl grpups. The calculated isoelectric point is 4.2 and the average charge per residue, 0.384. The computed partial specific volume and molecular volume were 0.734 g cm-3 and 34,000 .ANG.3, respectively. A study of the thermal stability of CaBP indicated that its immunoreactivity, high affinity binding of Ca and electrophoretic mobility were unchanged after a heat treatment of up to 80.degree., but declined precipitously between 80 and 90.degree.. Equilibrium dialysis studies revealed that Ca was bound exchangeably at 4 strong Ca binding sites with apparent intrinsic association constant, K(i), of 2 x 106 M-1 in 0.15 M KCl (pH 6.8). Based on published competitive binding data, the log K(i) for several divalent cations were calculated to be: Ca, 6.30; Cd, 5.10; Sr, 4.39-4.58; Mn, 4.37; **Zn**, 3.71; Ba, 3.18-3.24; Co, 2.84; Mg, 2.44. Binding affinity appears to be related to the crystal ionic radius of these various cations. Additional Ca binding appeared abruptly when the concentration of free Ca2+ reached 3 x 10-3M.

CT Medical Descriptors:
*chicken
*drug protein binding
*duodenum
*duodenum mucosa
*intestine
*intestine mucosa
*pharmacology
*protein binding
theoretical study
Drug Descriptors:
*amino acid
*calcium
*calcium binding protein
*colecalciferol
*enzyme
*ergocalciferol
***lipid**
*protein

RN (amino acid) 65072-01-7; (calcium) 7440-70-2; (colecalciferol) 1406-16-2, 67-97-0; (ergocalciferol) 50-14-6, 50809-47-7, 8042-78-2; (**lipid**) 66455-18-3; (protein) 67254-75-5

L6 ANSWER 19 OF 21 SCISEARCH COPYRIGHT 2003 ISI (R)
AN 97:369801 SCISEARCH
GA The Genuine Article (R) Number: WX710
TI Reactive oxygen species and nitric oxide in viral diseases
AU Peterhans E (Reprint)
CS UNIV BERN, INST VET VIROL, CH-3012 BERN, SWITZERLAND (Reprint)
CYA SWITZERLAND
SO BIOLOGICAL TRACE ELEMENT RESEARCH, (**JAN 1997**) Vol. 56, No. 1, pp. 107-116.
Publisher: HUMANA PRESS INC, 999 RIVERVIEW DRIVE SUITE 208, TOTOWA, NJ 07512.
ISSN: 0163-4984.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 75
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
SO BIOLOGICAL TRACE ELEMENT RESEARCH, (**JAN 1997**) Vol. 56, No. 1, pp. 107-116.
Publisher: HUMANA PRESS INC, 999 RIVERVIEW DRIVE SUITE 208, TOTOWA, NJ

07512.
ISSN: 0163-4984.

AB Metabolites derived from superoxide (O-2(.-)) and nitric oxide (NO .) play an important role in antimicrobial and antitumoral defense, but may also harm the host. Low levels of such metabolites can also facilitate viral replication because of their mitogenic effects on cells. Most viruses grow better in proliferating cells, and indeed, many viruses induce in their host cell changes similar to those seen early after treatment with mitogenic lectins. influenza and paramyxoviruses activate in phagocytes the generation of superoxide by a mechanism involving the interaction between the viral surface **glycoproteins** and the phagocyte's plasma membrane. Interestingly, viruses that activate this host defense mechanism are toxic when injected in the bloodstream of animals. Mice infected with influenza virus undergo oxidative stress. **Zn** addition, a wide array of cytokines are formed in the lung, contributing to the systemic effects of influenza. Oxidative stress is seen also in chronic viral infections, such as AIDS and viral hepatitis. Oxidant production in viral hepatitis may contribute to the emergence of hepatocellular carcinoma, a tumor seen in patients after years of chronic inflammation of the liver. Antioxidants and agents that downregulate proinflammatory cytokines and **lipid** mediators may be a useful complement to specific antiviral drugs in the therapy of viral diseases.

L6 ANSWER 20 OF 21 SCISEARCH COPYRIGHT 2003 ISI (R)
AN 95:138542 SCISEARCH
GA The Genuine Article (R) Number: QG848
TI INFLUENZA HEMAGGLUTININ-MEDIATED MEMBRANE-FUSION - INFLUENCE OF RECEPTOR-BINDING ON THE LAG PHASE PRECEDING FUSION
AU STEGMANN T (Reprint); BARTOLDUS I; ZUMBRUNN J
CS UNIV BASEL, BIOCTR, DEPT BIOPHYS CHEM, KLINGELBERGSTR 70, CH-4056 BASEL, SWITZERLAND (Reprint)
CYA SWITZERLAND
SO BIOCHEMISTRY, (**14 FEB 1995**) Vol. 34, No. 6, pp. 1825-1832.
ISSN: 0006-2960.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
SO BIOCHEMISTRY, (**14 FEB 1995**) Vol. 34, No. 6, pp. 1825-1832.
ISSN: 0006-2960.

AB Fusion of influenza virus with liposomes is triggered by low pH, resulting in a conformational change in the fusion protein (HA) and-the insertion of fusion peptides, from HA into the Liposomal membrane. Fusion does not take place immediately after insertion but is preceded by a lag phase, the duration of which, as we have found previously, depends on the presence of ganglioside receptors in the liposomal membrane [Stegmann, T., White, J. M., and Helenius, A. (1990) EMBO J. 9, 4231-4241]. Here we have investigated why that is the case. Surprisingly, the 2-4-fold shorter lag phase observed with phosphatidylcholine (PC)/phosphatidylethanolamine (PE)/ganglioside liposomes was ndt due to slower or more readily reversible binding of the virus to PC/PE liposomes lacking receptors. Nevertheless, using liposomes with various glycolipids as targets, it was found that specific HA-receptor interactions were required for a shorter lag, and not just the negative charge of the gangliosides, or the presence of ceramide **lipid** tails in the Liposomal membrane. Receptor binding also,did not facilitate the conformational change in HA. Surprisingly, however, it was found that after an incubation of the virus at low pH in the absence of target membranes at 0 degrees C for several minutes, the binding and fusion activity of virus using PC/PE liposomes,

but not PC/PE/ganglioside Liposomes as targets, was decreased. The population of virus that did still bind to and fuse with the PC/PE liposomes after low pH preincubation did so after a significantly increased lag time. Binding of virus to Liposomes without receptors is solely due to insertion of viral fusion peptides into the liposomal membrane, suggesting that the availability of fusion peptides is decreased after low pH preincubation. **Zn** accordance with this suggestion, if the Liposomal **Lipid** bilayers were in the gel phase, binding of virus to PC liposomes but not to PC/ganglioside liposomes was strongly inhibited, and the lag phase was about 9 times shorter for liposomes with receptors. Therefore, these results suggest that ganglioside receptors shorten the lag phase because they facilitate insertion of fusion peptides into the target membrane.

STP KeyWords Plus (R): PH-DEPENDENT FUSION; VIRUS HEMAGGLUTININ; CONFORMATIONAL CHANGE; SURFACE-DENSITY; KINETICS; INACTIVATION; LIPOSOMES; **GLYCOPROTEIN**; FLUORESCENCE; FIBROBLASTS

L6 ANSWER 21 OF 21 SCISEARCH COPYRIGHT 2003 ISI (R)
AN 94:374817 SCISEARCH
GA The Genuine Article (R) Number: NF487
TI ANTIGEN PRESENTATION BY MAJOR HISTOCOMPATIBILITY COMPLEX CLASS I-B MOLECULES
AU SHAWAR S M (Reprint); VYAS J M; RODGERS J R; RICH R R
CS BAYLOR COLL MED, DEPT MICROBIOL & IMMUNOL, HOUSTON, TX, 77030 (Reprint); BAYLOR COLL MED, DEPT MED, HOUSTON, TX, 77030
CYA USA
SO ANNUAL REVIEW OF IMMUNOLOGY, (**1994**) Vol. 12, pp. 839-880. ISSN: 0732-0582.
DT General Review; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 177
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
SO ANNUAL REVIEW OF IMMUNOLOGY, (**1994**) Vol. 12, pp. 839-880. ISSN: 0732-0582.
AB Class I-b genes constitute the majority of MHC class I loci. These monomorphic or oligomorphic molecules have been described in many organisms; they are best characterized in the mouse, which contains a substantial number of potentially intact genes. Two main characteristics differentiate class I-b from class I-a molecules: limited polymorphism and lower cell surface expression. These distinguishing features suggest possible generalizations regarding the evolution and function of this class. Additionally, class I-b proteins tend to have shorter cytoplasmic domains or in some cases may be secreted or may substitute a **lipid** anchor for the transmembrane domain. Some are also expressed in a limited distribution of cells or tissues.

STP KeyWords Plus (R): MHC CLASS-I; MATERNALLY TRANSMITTED ANTIGEN; CYTOTOXIC LYMPHOCYTES-T; HUMAN-PLASMA **ZN**-ALPHA-2-**GLYCOPROTEIN**; SEROLOGICALLY DEFINED LOCUS; INTESTINAL EPITHELIAL-CELLS; N-FORMYLATED PEPTIDES; HLA-G TRANSCRIPTS; LISTERIA-MONOCYTOGENES; TLA-REGION

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NEWS 4	Apr 09	ZDB will be removed from STN
NEWS 5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 9	Jun 03	New e-mail delivery for search results now available
NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
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NEWS 15	Jul 30	NETFIRST to be removed from STN
NEWS 16	Aug 08	CANCERLIT reload
NEWS 17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18	Aug 08	NTIS has been reloaded and enhanced
NEWS 19	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
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NEWS 21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22	Aug 26	Sequence searching in REGISTRY enhanced
NEWS 23	Sep 03	JAPIO has been reloaded and enhanced
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NEWS 25	Sep 16	CA Section Thesaurus available in CAPLUS and CA
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NEWS 27	Oct 21	EVENTLINE has been reloaded
NEWS 28	Oct 24	BEILSTEIN adds new search fields
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NEWS 30	Oct 25	MEDLINE SDI run of October 8, 2002
NEWS 31	Nov 18	DKILIT has been renamed APOLLIT
NEWS 32	Nov 25	More calculated properties added to REGISTRY
NEWS 33	Dec 02	TIBKAT will be removed from STN
NEWS 34	Dec 04	CSA files on STN
NEWS 35	Dec 17	PCTFULL now covers WP/PCT Applications from 1978 to date
NEWS 36	Dec 17	TOXCENTER enhanced with additional content
NEWS 37	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS 38	Dec 30	ISMEC no longer available
NEWS 39	Jan 13	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 40	Jan 21	NUTRACEUT offering one free connect hour in February 2003
NEWS 41	Jan 21	PHARMAML offering one free connect hour in February 2003
NEWS 42	Jan 29	Simultaneous left and right truncation added to COMPENDEX,

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ENERGY, INSPEC
NEWS 43 Feb 13 CANCERLIT is no longer being updated
NEWS 44 Feb 24 METADEX enhancements
NEWS 45 Feb 24 PCTGEN now available on STN
NEWS 46 Feb 24 TEMA now available on STN
NEWS 47 Feb 26 NTIS now allows simultaneous left and right truncation
NEWS 48 Feb 26 PCTFULL now contains images
NEWS 49 Mar 04 SDI PACKAGE for monthly delivery of multifile SDI results

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=> dup rem l1
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L2 112 DUP REM L1 (111 DUPLICATES REMOVED)

=> s l2 and py<=1998
1 FILES SEARCHED...
4 FILES SEARCHED...
L3 77 L2 AND PY<=1998

=> s l3 and mice
L4 17 L3 AND MICE

=> s l3 and (mice or adipocytes)
L5 18 L3 AND (MICE OR ADIPOCYTES)

=> s l5 and py<=1997
1 FILES SEARCHED...
4 FILES SEARCHED...
L6 15 L5 AND PY<=1997

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L6 ANSWER 1 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1998:50423 BIOSIS
DN PREV199800050423
TI Mechanism of depletion of liver glycogen in cancer cachexia.
AU Hirai, Kouzo; Ishiko, Osamu; Tisdale, Michael (1)
CS (1) Pharm. Sci. Inst., Aston Univ., Birmingham B3 7ET UK
SO Biochemical and Biophysical Research Communications, (**Dec. 8, 1997**) Vol. 241, No. 1, pp. 49-52.
ISSN: 0006-291X.
DT Article
LA English
SO Biochemical and Biophysical Research Communications, (**Dec. 8, 1997**) Vol. 241, No. 1, pp. 49-52.
ISSN: 0006-291X.
AB **Mice** transplanted with a cachexia-inducing colonic adenocarcinoma (MAC16) show a progressive decrease in liver glycogen in direct proportion to the loss of body weight. Such tumours elaborate a **lipid mobilizing factor** (LMF), which produces a dose-dependent stimulation, not only of adipocyte adenylate cyclase, but also of heptocyte adenylate cyclase in a GTP-dependent manner. These results suggest that LMF has the capacity to initiate hepatic glycogenolysis through an increase in cyclic AMP.
IT Major Concepts
Dental and Oral System (Ingestion and Assimilation); Tumor Biology
IT Parts, Structures, & Systems of Organisms
hepatocyte: digestive system; liver: digestive system
IT Diseases
cancer cachexia: disease-miscellaneous; colonic adenocarcinoma: digestive system disease, neoplastic disease
IT Chemicals & Biochemicals
cyclic AMP; enzymes; glycogen; **lipid mobilizing factor**; GTP

L6 ANSWER 2 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1996:334654 BIOSIS
DN PREV199699057010
TI Catabolic factors in cancer cachexia.
AU Tisdale, M. J. (1); McDevitt, T. M.; Todorov, P. T.; Cariuk, P.
CS (1) CRC Nutritional Biochemistry Res. Group, Pharmaceutical Science Inst., Aston Univ., Birmingham B4 7ET UK

09921880

SO In Vivo (Attiki), (1996) Vol. 10, No. 2, pp. 131-136.
ISSN: 0258-851X.
DT Article
LA English
SO In Vivo (Attiki), (1996) Vol. 10, No. 2, pp. 131-136.
ISSN: 0258-851X.
AB A **lipid mobilizing factor** has been purified from a cachexia-inducing mouse colon adenocarcinoma (MAC16) using a combination of ion exchange (Mono Q), exclusion (Superose) and reverse phase hydrophobic chromatography. The purification process led to a 3,500-fold increase in the specific activity. Serum from **mice** bearing the MAC16 tumour contained antibodies reactive with fractions containing lipid mobilizing activity and detectable as a 24kDa immunoreactive band on Western blotting. Serum from **mice** transplanted with a related tumour, MAC13, not producing cachexia, did not contain antibodies. A similar immunoreactive band was detectable in the urine of patients with cancer cachexia, but was absent from the urine of normal subjects. A monoclonal antibody produced by fusion of splenocytes from **mice** bearing the MAC16 tumour with mouse Balb/c myeloma cells attenuated the development of cachexia in **mice** transplanted with the MAC16 tumour and inhibited tumour growth. These results suggest that the Mr 24kDa antigen may be important in tumour growth and cachexia.
IT Miscellaneous Descriptors
CACHEXIA; CANCER; CATABOLIC FACTORS; **LIPID MOBILIZING FACTOR**; TUMOR GROWTH

L6 ANSWER 3 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1996:157926 BIOSIS
DN PREV199698730061
TI Induction of muscle protein degradation and weight loss by a tumor product.
AU Todorov, Penio T.; McDevitt, Trudi M.; Cariuk, Peter; Coles, Brian; Deacon, Melanie; Tisdale, Michael J. (1)
CS (1) Cancer Research Campaign Nutritional Biochemistry Research Group, Pharmaceutical Sci. Inst., Aston University, Aston Triangle Birmingham B4 7ET UK
SO Cancer Research, (1996) Vol. 56, No. 6, pp. 1256-1261.
ISSN: 0008-5472.
DT Article
LA English
SO Cancer Research, (1996) Vol. 56, No. 6, pp. 1256-1261.
ISSN: 0008-5472.
AB Splenocytes from **mice** bearing a cachexia-inducing tumor (MAC16) have been fused with mouse myeloma cells to produce hybridomas, which have been cloned to produce antibody reactive to a material which copurified with a **lipid-mobilizing factor** isolated from the same tumor. The monoclonal antibody has been used to investigate factors potentially involved in the development of cachexia. The major protein detectable by immunoprecipitation of a partially purified **lipid-mobilizing factor** was M-r 69,000, whereas Western blotting showed two bands of M-r 69,000 and M-r 24,000. Although the monoclonal antibody did not neutralize lipid-mobilizing activity in an in vitro assay, it did neutralize a serum factor capable of protein degradation in isolated gastrocnemius muscle. Affinity purification of MAC16 tumor homogenates using the monoclonal antibody yielded two immunoreactive bands of M-r 69,000 and M-r 24,000, which were further fractionated on a hydrophobic column (C-8). This material was capable of inducing tyrosine release from isolated gastrocnemius muscle, and the effect could be blocked with the monoclonal antibody. The two

immunoreactive bands from the hydrophobic column were capable of inducing weight loss in **mice**, whereas nonimmunoreactive fractions had no effect on body weight. The M-r 24,000 species had a unique amino acid sequence, whereas the M-r 69,000 species gave the same sequence as the M-r 24,000 material, together with that for albumin. The M-r 24,000 species contained carbohydrate, and lectin blotting showed a strong reaction with wheat germ and Erythrina crystagalli agglutinins. This suggests that the material is a glycoprotein or proteoglycan that shows strong binding affinity for albumin, possibly through the carbohydrate residues.

L6 ANSWER 4 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1995:223107 BIOSIS
DN PREV199598237407
TI Purification and characterization of a **lipid-mobilizing factor** associated with cachexia-inducing tumors in **mice** and humans.
AU McDevitt, Trudi M.; Todorov, Penio T.; Beck, Susan A.; Khan, Syrah H.; Tisdale, Michael J. (1)
CS (1) CRC Nutr. Biochem. Res. Group, Pharm. Sci. Inst., Aston Univ., Birmingham B4 7ET UK
SO Cancer Research, (1995) Vol. 55, No. 7, pp. 1458-1463. ISSN: 0008-5472.
DT Article
LA English
TI Purification and characterization of a **lipid-mobilizing factor** associated with cachexia-inducing tumors in **mice** and humans.
SO Cancer Research, (1995) Vol. 55, No. 7, pp. 1458-1463. ISSN: 0008-5472.
AB A scheme is described for the purification of a **lipid-mobilizing factor** from a cachexia-inducing murine tumor (MAC16) using a combination of ion exchange (Mono Q), exclusion (Superose), and hydrophobic (Cs) chromatography. This process yields an active material with an apparent molecular weight of 24,000 with an overall purification of 3,500 from the tumor homogenate and representing 0.005% of the total protein present. The material tends to aggregate to high molecular mass, is acidic (pI lt 4), and displays heterogeneity of charge as evidenced by a broad elution profile on ion exchange and exclusion chromatography and multiple peaks on hydrophobic columns. The purified material was heat and alkali (pH 10.4) labile and activity could be completely inhibited by sulfatase, suggesting that the negative charge could arise from sulfate residues. There was no evidence that the material possessed triglyceride lipase activity. Animals transplanted with the MAC16 tumor and with a delayed weight loss contained in their serum antibodies that recognized a M-r 24,000 band on Western blots. This material copurified with the **lipid-mobilizing factor**. Such antibodies were not present in the serum of **mice** transplanted with the MAC13 tumor, which does not induce cachexia, suggesting that the antibodies were directed to the induction of cachexia rather than the tumor itself. Urine from patients with cancer cachexia also contained a **lipid-mobilizing factor** which adhered to DEAE-cellulose and gave an apparent M-r of 24.000 by exclusion chromatography. Western blotting using serum from MAC16 tumor-bearing animals showed the presence of a band of M-r 24,000 in such fractions, which was not detected using serum from **mice** bearing the MAC13 tumor. This band was not present in Western blots of urine from normal subjects. The fact that serum from **mice** bearing the MAC16 tumor can detect the human lipid-mobilizing activity suggests a high degree of structural similarity between the two and raises the possibility that cachexia in humans may be caused by the same species

as in the mouse.

L6 ANSWER 5 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1991:410622 BIOSIS
DN BA92:77587
TI **LIPID MOBILIZING FACTORS** SPECIFICALLY ASSOCIATED WITH CANCER CACHEXIA.
AU BECK S A; TISDALE M J
CS CRC EXP. CHEMOTHERAPY GROUP, PHARM. SCI. INST., ASTON UNIV., BIRMINGHAM B4 7ET, UK.
SO BR J CANCER, (1991) 63 (6), 846-850.
CODEN: BJCAAI. ISSN: 0007-0920.
FS BA; OLD
LA English
TI **LIPID MOBILIZING FACTORS** SPECIFICALLY ASSOCIATED WITH CANCER CACHEXIA.
SO BR J CANCER, (1991) 63 (6), 846-850.
CODEN: BJCAAI. ISSN: 0007-0920.
AB Both urine and plasma from **mice** and humans with cancer cachexia have been shown to contain higher levels of lipid mobilizing activity than normal controls, even after acute starvation. There was no significant increase in the urinary lipid mobilizing activity of either **mice** or humans after acute starvation, suggesting that the material in the cachectic situation was probably not due to an elevation of hormones normally associated with the catabolic state in starvation. Further characterization of the lipid mobilizing activity in the urine of cachectic **mice** using Sephadex G50 exclusion chromatography showed four distinct peaks of activity of apparent molecular weights of > 20, 3, 1.5 and < 0.7 kDa. No comparable peaks of activity were found in the urine of a nontumor-bearing mouse. The high molecular weight activity was probably formed by aggregation of low molecular weight material, since treatment with 0.5 M NaCl caused dissociation to material with a broad spectrum of molecular weights between 3 and 0.7 kDa. Lipolytic species of similar molecular weights were also found in the urine of cachectic cancer patients, but not in normal urine even after 24 h starvation. The lipid mobilizing species may be responsible for catabolism of host adipose tissue in the cachectic state.

L6 ANSWER 6 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1991:116588 BIOSIS
DN BA91:63978
TI INHIBITION OF TUMOR-INDUCED LIPOLYSIS IN-VITRO AND CACHEXIA AND TUMOR GROWTH IN-VIVO BY EICOSAPENTAENOIC ACID.
AU TISDALE M; BECK S A
CS CRC EXP. CHEMOTHERAPY GROUP, PHARM. SCI. INST., ASTON UNIV., BIRMINGHAM B4 7ET, UK.
SO BIOCHEM PHARMACOL, (1991) 41 (1), 103-108.
CODEN: BCPCA6. ISSN: 0006-2952.
FS BA; OLD
LA English
SO BIOCHEM PHARMACOL, (1991) 41 (1), 103-108.
CODEN: BCPCA6. ISSN: 0006-2952.
AB Stimulation of lipolysis in murine **adipocytes** in response to a **lipid-mobilizing factor** prduced by a cachexia-inducing murine adenocarcinoma was inhibited by eicosapentaenoic acid (EPA) with a Ki value of 104 .mu.M. The inhibitory effect was strictly structurally specific, since other related fatty acids of both the (n-3) and (n-6) series were ineffective as inhibitors of the lipolytic process. Induction of lipolysis by both salbutamol and ACTH was also inhibited by EPA, suggesting that the effect is exerted on a step central

to the process of lipolysis. Lipolysis induced with the tumor **lipid-mobilizing factor** was associated with a prolonged elevation of the intracellular level of cyclic AMP in **adipocytes**, in contrast with ACTH and salbutamol. The elevation of adipocyte cycle AMP in response to the tumour **lipid-mobilizing factor** and lipolytic hormones was inhibited by EPA. In vivo, administration of pure EPA to weight losing **mice** bearing the MAC16 adenocarcinoma completely prevented weight loss and tumour growth weight. In contrast both the other (n-3) fatty acid present in fish oil, docosahexaenoic acid (DHA), and linoleic acid were ineffective in inhibiting weight loss or the growth of the MAC16 tumour. This suggests that inhibition of tumour lipolytic activity accounts for the anticachectic effect of EPA, and that a correlation may exist between the inhibition of cachexia and the inhibition of tumour growth.

L6 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1990:241203 BIOSIS
DN BA89:128156
TI REDUCED SUPPRESSION OF PLASMA SATURATED FATTY ACID MOBILIZATION AND OXIDATION BY FEEDING IN LYMPHOMA-BEARING **MICE**.
AU KANNAN R; GAN-ELEPANO M; BAKER N
CS JOHN MUIR CANCER AGING RESEARCH INST., 2055 NORTH BROADWAY, WALNUT CREEK, CALIF. 94596.
SO CANCER RES, (1990) 50 (8), 2221-2227.
CODEN: CNREA8. ISSN: 0008-5472.
FS BA; OLD
LA English
TI REDUCED SUPPRESSION OF PLASMA SATURATED FATTY ACID MOBILIZATION AND OXIDATION BY FEEDING IN LYMPHOMA-BEARING **MICE**.
SO CANCER RES, (1990) 50 (8), 2221-2227.
CODEN: CNREA8. ISSN: 0008-5472.
AB Lymphoma-bearing **mice** have a circulating **lipid-mobilizing factor**, but increased plasma free fatty acid (FFA) turnover has not been demonstrable in earlier studies using postabsorptive tumor-bearing **mice**. We hypothesized that FFA mobilization in lymphoma-bearing **mice** is only elevated in fed **mice** and may best be observed at night (dark, reversed light cycle). AKR **mice** with early and advanced tumors (106 SL-3 lymphoma cells, i.p.) and controls were fed ad libitum (reversed light cycle, dark) or fasted 4 h (daylight, regular cycle) given injections of [14C]bicarbonate or [1-14C]palmitate-mouse serum albumin, i.v., and plasma [14C]FFA disappearance and/or breath 14CO2 were monitored. Plasma FFA mobilization, estimated by multicompartmental analysis (SAAM) of the oxidation rate was lower in fasted **mice** with advanced tumors [tumor, 9.5 .+-. 6.0% (%SE); controls, 14 .+-. 4.4% .mu.g-atoms fatty acid-carbon/min/30 g body weight, n = 3 to 6 **mice**/time point/group]. Feeding reduced these rates 90% in control **mice** and 53% in **mice** with early tumors, but only 14% in **mice** with advanced tumors. Plasma FFA fractional catabolic rates were 2.5 times faster in fed **mice** with advanced tumors than in controls. Diminished suppression of fatty acid mobilization in fed tumor-bearing **mice** (at night) probably accounts partially for the body fat loss.

L6 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1990:91155 BIOSIS
DN BA89:50506
TI TURNOVER AND FATE OF PLASMA FREE FATTY ACIDS IN BRIEFLY-FASTED LYMPHOMA-BEARING **MICE**.
AU BAKER N; GAN-ELEPANO M; GUTHRIE B A; MEAD J F
CS JOHN MUIR CANCER AND AGING RES. INST., 2055 N. BROADWAY, WALNUT CREEK,

CALIF. 94596.
SO LIPIDS, (1989) 24 (12), 1028-1034.
CODEN: LPDSAP. ISSN: 0024-4201.
FS BA; OLD
LA English
TI TURNOVER AND FATE OF PLASMA FREE FATTY ACIDS IN BRIEFLY-FASTED LYMPHOMA-BEARING **MICE**.
SO LIPIDS, (1989) 24 (12), 1028-1034.
CODEN: LPDSAP. ISSN: 0024-4201.
AB Body fat loss during tumor growth may be due to increased mobilization of adipose triglycerides. Earlier work from this laboratory suggested that (i) lymphoma-bearing AKR **mice** have a circulating **lipid mobilizing factor** (LMF) which caused body fat loss during cancer growth; that (ii) fatty acids (FA) mobilized in these tumor-bearing (TB) **mice** were not oxidized to CO2 as in starved **mice** that lose their body fat; and that (iii) instead, the mobilized FA were sequestered by the lymphoma. We tested these hypotheses by injecting [114C]palmitate-albumin into lymphoma-bearing and control **mice**. We measured turnover of plasma FFA for 24 hr and predicted the cumulative conversion of tracer into breath 14CO2 (at 85 min) in the TB **mice**. Plasma FFA were mobilized more slowly in briefly fasted tumor-bearing **mice** than in controls with the same plasma FFA pool sizes. The fractional catabolic rate (FCR) (min-1) of plasma FFA turnover in both groups decreased during the night when the **mice** ate: postabsorptive controls, 1.07 (.+-. 5.6%); fed controls, 0.25 (.+-. 13%); postabsorptive TB, 0.53 (.+-. 4.6%); fed TB, 0.29 (.+-. 7.3%). Virtually all of the plasma FFA irreversible disposal in TB **mice** was accounted for as breath 14CO2 (30 to 40% I.D.), not as tumor lipids (1.1 .+-. 0.22% I.D.). Thus, FFA oxidation to CO2 is the major fate of plasma FFA turnover in TB **mice**, and sequestration of FFA (palmitate) by tumor cells is a quantitatively minor process. The putative circulating LMF did not cause increased FFA mobilization in these lymphoma-bearing **mice** in the post-absorptive state.
IT Miscellaneous Descriptors
BODY FAT LOSS **LIPID MOBILIZING FACTOR**
OXIDATION TUMOR SEQUESTRATION

L6 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1980:218062 BIOSIS
DN BA70:10558
TI A **LIPID MOBILIZING FACTOR** IN SERUM OF TUMOR BEARING **MICE**.
AU KITADA S; HAYS E F; MEAD J F
CS LAB. NUCL. MED. RADIAT. BIOL., UNIV. CALIF., 900 VETERAN AVE., LOS ANGELES, CALIF. 90024, USA.
SO LIPIDS, (1980) 15 (3), 168-174.
CODEN: LPDSAP. ISSN: 0024-4201.
FS BA; OLD
LA English
TI A **LIPID MOBILIZING FACTOR** IN SERUM OF TUMOR BEARING **MICE**.
SO LIPIDS, (1980) 15 (3), 168-174.
CODEN: LPDSAP. ISSN: 0024-4201.
AB There is considerable evidence that the growing tumor requires a source of unsaturated fatty acids, but the nature of this source and the mechanism of mobilizing the fatty acids from it are obscure. AKR **mice** with implanted adipose tissue labeled with 1-14C linoleic acid were used. In the normal, fed mouse, fat is mobilized slowly and appears largely as respiratory CO2, following oxidation. In the normal, fasted mouse, fat is mobilized rapidly and appears largely as respiratory CO2. In the

tumor-bearing, fed mouse, fat is mobilized rapidly and appears largely in the tumor. The serum from tumor-bearing **mice**, when injected into normal **mice**, produces an immediate massive fat mobilization that does not respond to feeding, whereas the serum from normal, fed **mice** does not. A mobilizing factor of unknown nature is present in the serum of tumor-bearing AKR **mice**.

L6 ANSWER 10 OF 15 MEDLINE
AN 97002878 MEDLINE
DN 97002878 PubMed ID: 8850217
TI Inhibition of lipolysis and muscle protein degradation by EPA in cancer cachexia.
AU Tisdale M J
CS Pharmaceutical Sciences Institute, Aston University, Birmingham, United Kingdom.
SO NUTRITION, **(1996 Jan)** 12 (1 Suppl) S31-3.
Journal code: 8802712. ISSN: 0899-9007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
ED Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961206
SO NUTRITION, **(1996 Jan)** 12 (1 Suppl) S31-3.
Journal code: 8802712. ISSN: 0899-9007.
AB Depletion of muscle and adipose tissue in cancer cachexia appears to arise not only from decreased food intake but also from the production of catabolic factors by certain tumours. Experiments with the cachexia-inducing MAC16 tumour in **mice** showed that when part of the carbohydrate calories were replaced by fish oil, host body weight loss was inhibited. The effect occurred without an alteration of either the total calorie consumption or nitrogen intake. Instead, one of the polyunsaturated fatty acids (PUFA) in fish oil, eicosapentaenoic acid (EPA), was found directly to inhibit tumour-induced lipolysis. The effect was structurally specific, as two related PUFA, docosahexaenoic acid (DHA) and gamma-linolenic acid (GLA), were without effect. The antilipolytic effect of EPA arose from an inhibition of the elevation of cyclic AMP in **adipocytes** in response to the **lipid mobilizing factor**. The increased protein degradation in the skeletal muscle of cachectic animals was also inhibited by EPA. This effect was due to the inhibition of the rise in muscle prostaglandin E2 in response to a tumour-produced proteolytic factor by EPA. Thus, reversal of cachexia by EPA in this mouse model results from its capacity to interfere with tumour-produced catabolic factors. Similar factors have been detected in human cancer cachexia.
CT Check Tags: Animal; Support, Non-U.S. Gov't
*5,8,11,14,17-Eicosapentaenoic Acid: TU, therapeutic use
*Adenocarcinoma: CO, complications
Cachexia: DT, drug therapy
Cachexia: ET, etiology
*Cachexia: ME, metabolism
*Colonic Neoplasms: CO, complications
Dinoprostone: ME, metabolism
Fish Oils: TU, therapeutic use
*Lipolysis: DE, drug effects
Mice
*Muscle Proteins: ME, metabolism
Neoplasm Transplantation

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Tumor Cells, Cultured

L6 ANSWER 11 OF 15 MEDLINE
AN 68275643 MEDLINE
DN 68275643 PubMed ID: 4172515
TI The lipid-mobilizing effect of some pituitary gland preparations. 3. A purified human pituitary **lipid-mobilizing factor** (LMF) with hyperglycaemic activity.
AU Trygstad O
SO ACTA ENDOCRINOLOGICA, **(1968 Jun)** 58 (2) 277-94.
Journal code: 0370312. ISSN: 0001-5598.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196807
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19680730
TI The lipid-mobilizing effect of some pituitary gland preparations. 3. A purified human pituitary **lipid-mobilizing factor** (LMF) with hyperglycaemic activity.
SO ACTA ENDOCRINOLOGICA, **(1968 Jun)** 58 (2) 277-94.
Journal code: 0370312. ISSN: 0001-5598.
CT Check Tags: Animal; Human; In Vitro
Adipose Tissue: DE, drug effects
*Adipose Tissue: ME, metabolism
Biological Assay
Chromatography, Gel
Dactinomycin: PD, pharmacology
Electrophoresis, Disc
Epinephrine: PD, pharmacology
Fatty Acids, Nonesterified: BL, blood
Hyperglycemia: CI, chemically induced
Hypocalcemia: CI, chemically induced
Lipids: ME, metabolism
*Lipotropic Agents: PD, pharmacology
Lipotropin: PD, pharmacology
Mice
Molecular Weight
Pituitary Gland: AN, analysis
*Pituitary Hormones, Anterior: PD, pharmacology
Rabbits
Rats
Sodium Chloride
Somatotropin: PD, pharmacology
Spectrophotometry
Stimulation, Chemical
Ultraviolet Rays

L6 ANSWER 12 OF 15 MEDLINE
AN 68163213 MEDLINE
DN 68163213 PubMed ID: 4295980
TI The lipid-mobilizing effect of some pituitary gland perparations. II. Preparation of a human pituitary **lipid-mobilizing factor** (LMF) with hypocalcaemic and hyperglycaemic effects in rabbits.
AU Trygstad O
SO ACTA ENDOCRINOLOGICA, **(1968 Jan)** 57 (1) 81-108.
Journal code: 0370312. ISSN: 0001-5598.

09921880

CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196805
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19680509
TI The lipid-mobilizing effect of some pituitary gland perparations. II. Preparation of a human pituitary **lipid-mobilizing factor** (LMF) with hypocalcaemic and hyperglycaemic effects in rabbits.
SO ACTA ENDOCRINOLOGICA, **(1968 Jan)** 57 (1) 81-108.
Journal code: 0370312. ISSN: 0001-5598.
CT Check Tags: Animal; Female; Human; In Vitro; Male
Acrylic Resins
Adipose Tissue: ME, metabolism
Biological Assay
Cellulose
Chromatography, Gel
Chromatography, Ion Exchange
Corticotropin: AN, analysis
Electrophoresis
Fatty Acids, Nonesterified: BL, blood
Gels
*Hyperglycemia: CI, chemically induced
*Hypocalcemia: CI, chemically induced
Lipoproteins: AN, analysis
MSH: AN, analysis
Mice
Molecular Weight
*Pituitary Gland: AN, analysis
*Pituitary Hormones, Anterior: AN, analysis
Prolactin: AN, analysis
Rabbits

L6 ANSWER 13 OF 15 MEDLINE
AN 66069060 MEDLINE
DN 66069060 PubMed ID: 5853868
TI [Investigations on direct acting lipid mobilizers of the organism. (II). Investigations on the physico-chemical properties of a **lipid-mobilizing factor** isolated from human blood].
Untersuchungen uber direkt wirkende Lipoidmobilisatoren des Organismus. (II). Untersuchungen uber die physiko-chimischen Eigneschaften eines aus menschlichem Blut isolierbaren lipoidmobilisierenden Faktors.
AU Kadas L; Nagy D
SO ENDOKRINOLOGIE, **(1965 Jun)** 48 (1) 8-14.
Journal code: 0370675. ISSN: 0013-7251.
CY GERMANY, EAST: German Democratic Republic
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 196603
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19660319
TI [Investigations on direct acting lipid mobilizers of the organism. (II). Investigations on the physico-chemical properties of a **lipid-mobilizing factor** isolated from human blood].
Untersuchungen uber direkt wirkende Lipoidmobilisatoren des Organismus.

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(II). Untersuchungen uber die physiko-chimischen Eigneschaften eines aus menschlichem Blut isolierbaren lipoidmobilisierenden Faktors.
SO ENDOKRINOLOGIE, **(1965 Jun)** 48 (1) 8-14.
Journal code: 0370675. ISSN: 0013-7251.
CT Check Tags: Animal; Human; In Vitro
Chemistry, Physical
Cholesterol: ME, metabolism
Chromatography, Paper
Cortisone: PD, pharmacology
Electrophoresis
Fasting
Hypothalamus
Ion Exchange
*Lipids: ME, metabolism
Mice
Pituitary Gland, Posterior

L6 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2003 ACS
AN 1965:419255 CAPLUS
DN 63:19255
OREF 63:3440a-b
TI Fat-mobilizing substance in the urine of patients with diabetes and with pituitary diseases and the effect of insulin on its action
AU Goth, A.; Hegedus, G.
CS Janos Hosp., Budapest, Hung.
SO Experientia (**1965**), 21(5), 277-8
DT Journal
LA English
SO Experientia (**1965**), 21(5), 277-8
AB Exts., prepd. by the method of Chalmers, et al. (CA 54, 25155a) from urine of normal persons on a restricted caloric diet or of untreated diabetics and injected on alternate days into **mice**, caused wt. loss, decreased blood sugar, and increased blood and liver lipid concn. Exts. from urine of normal persons on adequate caloric intake or from diabetics controlled with insulin were inactive. Exts. from urine of 2 acromegalics on adequate diet were active, while those from a patient with Sheehan's syndrome were inactive.
IT Urine
(**lipid-mobilizing factor** in, in acromegaly and diabetes, insulin effect on)

L6 ANSWER 15 OF 15 SCISEARCH COPYRIGHT 2003 ISI (R)
AN 97:660942 SCISEARCH
GA The Genuine Article (R) Number: XT920
TI Induction of cachexia in **mice** by a product isolated from the urine of cachectic cancer patients
AU Cariuk P; Lorite M J; Todorov P T; Field W N; Wigmore S J; Tisdale M J (Reprint)
CS ASTON UNIV, INST PHARMACEUT SCI, BIRMINGHAM B4 7ET, W MIDLANDS, ENGLAND (Reprint); ASTON UNIV, INST PHARMACEUT SCI, BIRMINGHAM B4 7ET, W MIDLANDS, ENGLAND; UNIV EDINBURGH, ROYAL INFIRM, DEPT SURG, EDINBURGH EH3 9YW, MIDLOTHIAN, SCOTLAND
CYA ENGLAND; SCOTLAND
SO BRITISH JOURNAL OF CANCER, (**28 AUG 1997**) Vol. 76, No. 5, pp. 606-613.
Publisher: CHURCHILL LIVINGSTONE, JOURNAL PRODUCTION DEPT, ROBERT STEVENSON HOUSE, 1-3 BAXTERS PLACE, LEITH WALK, EDINBURGH EH1 3AF, MIDLOTHIAN, SCOTLAND.
ISSN: 0007-0920.
DT Article; Journal

09921880

FS LIFE; CLIN
LA English
REC Reference Count: 37
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
TI Induction of cachexia in **mice** by a product isolated from the urine of cachectic cancer patients
SO BRITISH JOURNAL OF CANCER, (**28 AUG 1997**) Vol. 76, No. 5, pp. 606-613.
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AB Urine from cancer patients with weight loss showed the presence of an antigen of M(r)24000 detected with a monoclonal antibody formed by fusion of splenocytes from **mice** with cancer cachexia. The antigen was not present in the urine of normal subjects, patients with weight loss from conditions other than cancer or from cancer patients who were weight stable or with low weight loss (1 kg month(-1)). The antigen was present in the urine from subjects with carcinomas of the pancreas, breast, lung and ovary. The antigen was purified from urine using a combination of affinity chromatography with the mouse monoclonal antibody and reversed-phase high-performance liquid chromotography (HPLC). This procedure gave a 200000-fold purification of the protein over that in the original urine extract and the material isolated was homogeneous, as determined by silver staining of gels. The N-terminal amino acid sequence showed no homology with any of the recognized cytokines. Administration of this material to **mice** caused a significant ($P<0.005$) reduction in body weight when compared with a control group receiving material purified in the same way from the urine of a normal subject. Weight loss occurred without a reduction in food and water intake and was prevented by prior administration of the mouse monoclonal antibody. Body composition analysis showed a decrease in both fat and non-fat carcass mass without a change in water content. The effects on body composition were reversed in **mice** treated with the monoclonal antibody. There was a decrease in protein synthesis and an increase In degradation in skeletal muscle. Protein degradation was associated with an increased prostaglandin E-2 (PGE(2)) release. Both protein degradation and PGE(2) release were significantly reduced in **mice** pretreated with the monoclonal antibody. These results show that the material of M-r 24000 present in the urine of cachectic cancer patients is capable of producing a syndrome of cachexia in **mice**.
STP KeyWords Plus (R): TUMOR NECROSIS FACTOR; MUSCLE PROTEIN-DEGRADATION; **LIPID-MOBILIZING FACTOR**; WEIGHT-LOSS; ENERGY-EXPENDITURE; INTERLEUKIN-6; TURNOVER; CHILDREN; ANOREXIA; SKELETAL